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Original research

ASSOCIATION BETWEEN ADIPONECTIN CONCENTRATION AND METABOLIC SYNDROME IN INDIVIDUALS ATTENDING HEALTH CHECK - UPS AT HUE CENTRAL HOSPITAL

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ABSTRACT

Background: *Adiponectin, secreted by adipose tissue, is involved in the regulation of body weight. Dysregulation of adiponectin is associated with obesity, metabolic syndrome.*

Methods: *A cross-sectional study was conducted on 206 individuals aged over 18 years undergoing health check-ups at Hue Central Hospital from October 2025 to February 2026. Multivariate linear regression analysis was performed to evaluate the associations between adiponectin levels and study variables. Receiver operating characteristic (ROC) curve analysis was used to assess the discriminatory value of adiponectin for metabolic syndrome.*

Results: *The median serum adiponectin level was 7.64 (5.11 - 12.87). Serum adiponectin levels were significantly higher in females than in males [9.58 (6.26 - 13.94) vs. 5.59 (3.88 - 8.21), respectively; p < 0.001]. Multivariate linear regression analysis demonstrated that age, sex, BMI, and metabolic syndrome were independently associated with adiponectin levels. At a cut-off value of ≤ 5.26, adiponectin yielded a sensitivity of 40.5%, a specificity of 81.1%, and an AUC of 0.616 (p = 0.004) for metabolic syndrome.*

Conclusion: *Serum adiponectin levels were associated with components of metabolic syndrome in individuals undergoing health check-ups at Hue Central Hospital. Adiponectin levels were independently associated with age, sex, BMI, and metabolic syndrome.*

Keywords: *Adiponectin, sex, BMI, Mets.*

I. INTRODUCTION

Metabolic syndrome (MetS) is a cluster of interrelated metabolic abnormalities, including central obesity, dyslipidemia, hypertension, and impaired glucose metabolism, which collectively increase the risk of type 2 diabetes mellitus and cardiovascular disease [1]. The global prevalence of MetS has risen substantially in recent decades, largely driven by increasing rates of obesity and sedentary lifestyles, making it a major public health concern [2]. Despite extensive research, the underlying mechanisms linking these metabolic disturbances remain incompletely understood.

Adipose tissue is now recognized as an active endocrine organ that secretes a variety of bioactive molecules known as adipokines, which play key roles in metabolic regulation [3]. Among these, adiponectin is one of the most abundant adipokines and exerts insulin-sensitizing, anti-inflammatory, and anti-atherogenic effects [4]. Through its receptors, AdipoR1 and AdipoR2, adiponectin enhances fatty acid oxidation, improves glucose utilization, and suppresses hepatic gluconeogenesis, thereby contributing to metabolic homeostasis [5].

Paradoxically, circulating adiponectin levels are decreased in conditions characterized by

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insulin resistance, such as obesity, type 2 diabetes, and MetS [6]. Previous studies have consistently demonstrated inverse associations between adiponectin levels and key components of MetS, including abdominal obesity, hypertriglyceridemia, low HDL-cholesterol, and hyperglycemia [7]. In addition, adiponectin plays an important role in modulating inflammatory pathways, further supporting its protective effects against endothelial dysfunction and atherosclerosis [8].

Given these properties, adiponectin has been proposed not only as a biomarker of metabolic risk but also as a potential tool for risk stratification. Several studies have attempted to identify optimal cut-off values of adiponectin for predicting metabolic syndrome and insulin resistance using receiver operating characteristic (ROC) curve analysis, demonstrating moderate to good diagnostic performance [9]. Furthermore, regression-based approaches, including linear and logistic regression models, have been widely used to quantify the independent association between adiponectin and metabolic risk factors, even after adjustment for confounding variables such as age, sex, and body mass index (BMI) [7,10].

However, the relationship between adiponectin, metabolic syndrome, and insulin resistance may vary across populations due to differences in ethnicity, lifestyle, and clinical characteristics. In particular, data on adiponectin levels and their diagnostic and predictive value in Vietnamese populations remain limited. Therefore, evaluating adiponectin levels in conjunction with statistical approaches such as ROC analysis and regression modeling may provide more robust evidence for its clinical utility.

Accordingly, this study aimed to determine serum adiponectin levels in individuals undergoing health examinations at Hue Central Hospital and to evaluate the associations between adiponectin and age, sex, BMI, metabolic syndrome, and insulin resistance. In addition, we sought to assess the diagnostic performance of adiponectin and to identify optimal cut-off values for predicting metabolic syndrome and insulin resistance. This study aimed to determine serum adiponectin levels in individuals undergoing health check-ups at Hue Central Hospital, evaluate the associations between adiponectin and age, sex, BMI, metabolic syndrome, and insulin resistance, and assess the diagnostic performance of adiponectin, including identifying optimal cut-off values for predicting metabolic syndrome and insulin resistance.

II. SUBJECTS AND METHODS

2.1. Study population

Inclusion criteria: Age between 18 and 75 years; Attending Hue Central Hospital during the study period; Willingness to participate in the study (provided informed consent).

Exclusion criteria: Pregnancy or breastfeeding; Presence of severe acute conditions (e.g., severe infection, shock, recent myocardial infarction); Use of corticosteroids or medications affecting endocrine function; Participants receiving treatment for dyslipidemia or diabetes mellitus were excluded based on medical history taking, direct interviews, and review of medical records or current medications; Uncontrolled thyroid disorders

2.2. Study design

Study design: Cross-sectional descriptive study.

Setting: Outpatient Department, Hue Central Hospital, Department of General Internal Medicine and Endocrinology.

Study period: From October 2025 to February 2026.

Participants: Patients presenting for examination who met the study inclusion criteria.

Sample size calculation

The sample size was calculated using the following formula:

$$n=\frac{Z^2_{1-\alpha/2}.\delta^2}{d^2}$$

Where:

n: minimum required sample size

$Z_{1-\alpha/2}$: confidence coefficient; for a 95% confidence level, Z = 1.96

σ (SD): standard deviation of adiponectin concentration. According to the study by Vo Minh Phuong, σ = 5.06 µg/mL in the control group [11]

d: desired margin of error (absolute precision); d = 1 µg/mL

Calculated sample size: n ≈ 99

Minimum sample size: 99 participants

In this study, a total of 206 participants were included.

A convenience sampling method was used, including patients who attended health check-ups at the Outpatient Department of Hue Central Hospital.

2.3. Data collection procedures

Clinical assessments included measurements of body weight, height, waist circumference, and blood pressure.

Laboratory investigations included adiponectin, glucose, insulin, and lipid profile parameters (total cholesterol, LDL-C, HDL-C, and triglycerides).

2.4. Adiponectin measurement

Serum samples were stored at -80°C until analysis. Serum adiponectin concentrations were measured using an enzyme-linked immunosorbent assay (ELISA) kit (Diazyme) and quantified on a Beckman Coulter AU 5822 analyzer. The analytical measurement range of the assay was 1.12–40 µg/mL.

The intra-assay coefficient of variation (CV) ranged from 1.9% to 4.3%, and the inter-assay CV ranged from 0% to 4.9%. Adiponectin concentrations were expressed in µg/mL. Internal quality control procedures were implemented throughout all analytical processes to ensure data accuracy and reproducibility.

2.5. Diagnostic criteria for metabolic syndrome and insulin resistance

Diagnosis of metabolic syndrome (according to the 2009 Joint Interim Statement [JIS]) [1]

The criteria for metabolic syndrome include:

(1) Increased waist circumference, defined as ≥ 90 cm in men and ≥ 80 cm in women.

(2) Elevated triglycerides are defined as TG ≥ 150 mg/dL (≥ 1.7 mmol/L).

(3) Reduced HDL-cholesterol is defined as < 40 mg/dL (1.03 mmol/L), in men and < 50 mg/dL (1.29 mmol/L), in women.

(4) Elevated blood pressure is defined as blood pressure ≥ 130/85 mmHg.

(5) Elevated fasting glucose is defined as fasting plasma glucose ≥ 100 mg/dL (≥ 5.6 mmol/L).

Metabolic syndrome was diagnosed when ≥ 3 of the 5 criteria were present.

- The formula for calculating the HOMA-IR (Homeostatic Model Assessment of Insulin Resistance) index is:

$$\text{HOMA} - \text{IR} = \frac{\text{Fasting insulin } (\mu\text{U/mL}) \times \text{Fasting glucose (mmol/L)}}{22.5}$$

Where:

Fasting insulin: fasting serum insulin (µU/mL)

Fasting glucose: fasting blood glucose (mmol/L)

Insulin resistance was defined as HOMA-IR ≥ 2.5 [12].

- The QUICKI (Quantitative Insulin Sensitivity Check Index) was calculated using the following formula:

$$\text{QUICKI} = \frac{1}{\log[\text{Fasting insulin } (\mu\text{U/mL})] + \log[\text{Fasting glucose (mg/dL)}]}$$

Where:

Fasting insulin: fasting serum insulin (µU/mL)

Fasting glucose: fasting blood glucose (mg/dL)

A higher QUICKI value indicates greater insulin sensitivity.

2.6. Data Analysis

Statistical analyses were performed using SPSS version 26.0. Continuous variables were presented as mean ± standard deviation (SD) or median with interquartile range (IQR), as appropriate. Group comparisons were conducted using the independent samples t-test or one-way analysis of variance (ANOVA) for normally distributed data, and the Mann - Whitney U test for non - normally distributed data.

Correlations between adiponectin and BMI, insulin, and lipid parameters were assessed using Spearman correlation coefficients. A p-value < 0.05 was considered statistically significant. Multivariate linear regression analysis was performed to evaluate the associations between adiponectin levels and the study variables. Receiver operating characteristic (ROC) curve analysis was used to assess the discriminatory value of adiponectin for metabolic syndrome. The area under the ROC curve (AUC), optimal cutoff value, sensitivity, and specificity were determined.

2.7. Research Ethics

The study was conducted with the informed consent of all participants. All collected data were kept strictly confidential and used solely for research purposes. The study was approved by the Institutional Review Board of Hue University of Medicine and Pharmacy, Hue University (Approval No. H2025/670, dated October 8, 2025) and Ethics Committee in Biomedical Research of Hue Central Hospital.

III. RESULTS

The general characteristics of the study participants according to sex are presented in Table 1. Men had significantly higher waist circumference, systolic and diastolic blood pressure, and triglyceride levels than women. In contrast, HDL-C and adiponectin levels were significantly lower in men than in women ($p < 0.001$).

The correlations between adiponectin levels and metabolic syndrome risk factors are presented in Table 2. Adiponectin levels were negatively correlated with waist circumference, systolic blood pressure, BMI, total cholesterol, triglycerides, fasting blood glucose, insulin levels, and HOMA-IR (all $p < 0.05$). Conversely, adiponectin levels were positively correlated with age, HDL-C, and QUICKI (all $p < 0.001$) (Table 1).

Table 1: General characteristics of the study participants according to sex

Parameters	All (n=206)	Men (n=69)	Women (n=137)	p
Age (y)	50.2 ± 11.4	49.5 ± 11.7	50.5 ± 11.3	0.318
Waist circumference (cm)	84 (80 - 90)	90 (83 - 92)	82 (80 - 86)	< 0.001
BMI (kg/m^2)	22.9 (21.2 - 24.8)	23.56 (22.0 - 24.9)	22.7 (21.0 - 24.5)	0.072
Systolic BP (mmHg)	120 (120 - 130)	120 (120.00 - 130)	120 (110.00 - 130)	0.005
Diastolic BP (mmHg)	80 (70 - 80)	80 (70 - 80)	70 (70 - 80)	0.004
TG (mmol/L)	1.48 (0.99 - 2.17)	1.68 (1.30 - 3.46)	1.40 (0.91 - 1.99)	0.001
HDL cholesterol (mmol/L)	1.35 (1.15 - 1.58)	1.20 (1.05 - 1.44)	1.42 (1.23 - 1.66)	< 0.001
FBG (mmol/L)	5.40 (5.10 - 5.80)	5.50 (5.20 - 5.84)	5.37 (5.05 - 5.80)	0.091
Insulin (µU/dL)	6.50 (4.47 - 9.60)	6.65 (4.63 - 9.66)	6.28 (4.43 - 9.73)	0.969
HOMA-IR	1.60 (1.06 - 2.40)	1.59 (1.13 - 2.40)	1.60 (1.06 - 2.38)	0.904
QUICKI	0.36 (0.33 - 0.38)	0.35 (0.33 - 0.37)	0.36 (0.33 - 0.38)	0.730
Adiponectin	7.64 (5.11 - 12.87)	5.59 (3.88 - 8.21)	9.58 (6.26 - 13.94)	< 0.001
Mets	74 (35.9%)	23 (33.3%)	51 (37.2%)	0.583
IR	45 (21.8%)	15 (21.7%)	30 (21.9%)	0.979

Values are expressed as means ± SD or number (%) or Median (lower quartile-upper quartile).

SD, standard deviation; BMI, body mass index; TG, triglycerides; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; FBG, fasting blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin sensitivity check index; MetS, metabolic syndrome; IR, insulin resistance.

Multivariate linear regression analysis of the associations between adiponectin levels and the study variables (Table 3) demonstrated that age, female sex, BMI, and metabolic syndrome were independently associated with adiponectin levels ($p < 0.01$) (Table 2).

Table 2: Nonparametric correlations analysis of adiponectin levels with study parameters

Parameters	rho	p-value
Age (y)	0.30	< 0.001
Waist circumference (cm)	-0.21	0.002
BMI (kg/m^2)	-0.26	< 0.001

Parameters	rho	p-value
Systolic BP (mmHg)	-0.15	0.027
Diastolic BP (mmHg)	-0.10	0.135
TG (mmol/L)	-0.35	< 0.001
HDL cholesterol (mmol/L)	0.43	< 0.001
FBG (mmol/L)	-0.17	0.012
Insulin (µU/dL)	-0.27	< 0.001
HOMA-IR	-0.26	< 0.001
QUICKI	0.31	< 0.001

rho: Spearman correlation coefficient; Y, years; BMI, body mass index; TG, triglycerides; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; FBG, fasting blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin sensitivity check index.

Table 3: Multivariate linear regression analysis assessing the association between adiponectin levels and study parameters

Independent variable	B	95% CI B		β	p
		Lower	Upper		
Age (y)	0.12	0.06	0.19	0.25	< 0.001
Female	3.38	1.77	4.99	0.28	< 0.001
BMI	-0.40	-0.70	-0.09	-0.21	0.011
Waist circumference	0.07	-0.06	0.20	0.09	0.303
Mets	-2.70	-4.37	-1.03	-0.23	0.002
IR	-0.25	-2.18	1.68	-0.02	0.800

Y, year; BMI, body mass index; Mets, metabolic syndrome; IR, insulin resistance.

At a cut-off value of ≤ 5.26, adiponectin showed a sensitivity of 40.5%, a specificity of 81.1%, and an AUC of 0.616 (p = 0.004) for metabolic syndrome. Although adiponectin was significantly associated with metabolic syndrome, its discriminatory value as a standalone marker remained limited, Figure 1.

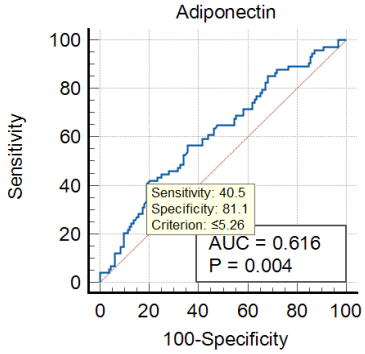

Figure 1: Optimal cut-off value of adiponectin for predicting metabolic syndrome

IV. Discussion

In this study, we found that adiponectin is associated with MetS and most of the MetS components. Adiponectin is a multifunctional protein with pleiotropic insulin-sensitizing effects and is considered a key molecule in the pathogenesis of MetS [20-22].

Our results showed that adiponectin levels were significantly higher in females than in males, which is consistent with many previous studies both domestically and internationally. In the study by Vo Minh Phuong, adiponectin levels were higher in females than in males in both the overweight/obese group (14.84 ± 3.44 vs. 4.85 ± 1.37) and the control group (8.92 ± 5.24 vs. 4.22 ± 3.82) [11]. This difference may be explained by the greater proportion of subcutaneous fat in females, the influence of estrogen on adiponectin expression, and sex-related differences in fat distribution.

Our study demonstrated negative correlations between adiponectin levels and insulin as well as HOMA-IR, while a positive correlation was observed between adiponectin and QUICKI. However, in the multivariate regression model, insulin resistance was not an independent predictor of adiponectin levels. Similar findings have been reported in previous studies. Ding Y.S. demonstrated a weak inverse correlation between adiponectin levels and HOMA-IR ($r = -0.112$, $p < 0.001$) [10].

Our study showed that adiponectin was inversely correlated with waist circumference, systolic blood pressure, triglycerides, and glucose, and positively correlated with HDL-C. These findings are consistent with previous studies across different populations. In the study by Vo Minh Phuong, adiponectin was positively correlated with waist circumference, hip circumference, and BMI ($p < 0.01$–0.05) [11]. The study by Ding Y.S. showed weak inverse correlations between adiponectin levels and waist circumference ($r = -0.212$, $p < 0.001$), systolic blood pressure ($r = -0.121$, $p < 0.001$), and triglycerides ($r = -0.016$, $p < 0.001$) [10].

According to Koh et al., adiponectin levels were inversely correlated with waist circumference ($r = -0.22$), triglycerides ($r = -0.23$), blood pressure ($r = -0.06$), fasting blood glucose ($r = -0.13$), HOMA-IR ($r = -0.22$), and CRP ($r = -0.03$), while showing a positive correlation with HDL cholesterol ($r = 0.23$) [17]. According to von Frankenberg et al., adiponectin levels were positively correlated with HDL-cholesterol ($r = 0.452$, $p < 0.001$) and negatively correlated with waist circumference ($r = -0.269$, $p < 0.001$), fasting glucose ($r = -0.289$, $p = 0.001$), and triglycerides ($r = -0.252$, $p < 0.001$). No significant correlations were observed between adiponectin levels and systolic blood pressure ($r = -0.135$, $p = 0.081$) or diastolic blood pressure ($r = -0.143$, $p = 0.066$) [18].

Our multivariate regression analysis showed that adiponectin was independently associated with metabolic syndrome. Similarly, Kim J.Y. reported that higher adiponectin levels were a protective factor against MetS in both sexes. Median adiponectin levels were lower in patients with MetS compared to those without MetS in both men (7.09 vs. 8.63, $p < 0.001$) and women (10.96 vs. 12.16, $p < 0.001$)[10].

As shown in Figure 1, at a cut-off value of ≤ 5.26, adiponectin demonstrated a sensitivity of 40.5%, a specificity of 81.1%, and an AUC of 0.616 ($p = 0.004$) for identifying metabolic syndrome. Despite its significant association with metabolic syndrome, adiponectin showed limited diagnostic performance when used as an isolated biomarker.

According to Liu Z. et al., adiponectin demonstrated moderate accuracy in identifying metabolic syndrome, with an area under the curve (AUC) of 0.81 (95% CI: 0.77–0.84) [15]. Similarly, Ding S. et al. reported that the AUC of adiponectin for detecting MetS was 0.639 based on the IDF criteria and 0.715 based on the ATP III criteria [16].

Study Limitations: Our study was a single-center study with a limited sample size, which may not be representative of the general population. As a cross-sectional descriptive study, it lacked longitudinal follow-up; therefore, causal relationships could not be established. In addition, we did not include patients receiving treatment. Untreated patients are less likely to have established comorbidities such as diabetes mellitus, chronic kidney disease, or cardiovascular disease, which limited our ability to stratify analyses according to underlying conditions (e.g., diabetes, hypertension).

V. CONCLUSION

Serum adiponectin levels were significantly associated with metabolic syndrome, insulin resistance, age, sex, and BMI in individuals undergoing health check-ups at Hue Central Hospital.

Conflict of interest statement

The authors declare that there are no conflicts of interest related to this study, the authorship, or the publication of this article.

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